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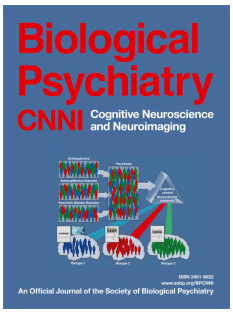

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Abstract

Background: The majority of reward learning neuroimaging studies have not focussed on the motivational aspects of behaviour, such as the inherent value placed on choice itself. The experience and affective value of personal control may have particular relevance for psychiatric disorders including depression.

Methods/Design: In this study, we adapted an fMRI reward task that probed the value placed on exerting control over one's decisions, termed 'choice value', in 122 healthy participants. We examined activation associated with choice value; personally-chosen versus passively-received rewards; and reinforcement learning metrics such as prediction error. Relationships were tested between measures of motivational orientation (categorised as Autonomy, Control and Impersonal), and subclinical depressive symptomatology.

Results: Anticipating personal choice activated left insula, cingulate, right inferior frontal cortex and ventral striatum ($P<0.05$ FWE-corrected). Ventral striatal activations to choice were diminished in those with subclinical depressive symptomatology. Personally-chosen rewards were associated with greater activation of the insula/IFG, cingulate cortex, hippocampus, thalamus and substantia nigra compared to rewards that were passively received. In people who felt little control over their own behaviour (Impersonal orientation), prediction error signals in nucleus accumbens were stronger during passive trials.

Discussion: Previous finding regarding personal choice have been verified, and taken forward through the use of both reinforcement learning models, and correlations with psychopathology. Personal choice has an impact on the extended reward network, potentially allowing these clinically-important areas to be addressed in ways more relevant to personality styles, self-esteem and symptoms such as motivational anhedonia.

Keywords: depression, imaging, locus of causality, perceived control, reward learnng, value of choice

Introduction

Disruption in motivation and reward processing are key elements of many psychiatric disorders, including anhedonia in major depressive disorder (MDD), negative symptoms in schizophrenia, and mania in bipolar disorder (1).

Standard reward tasks (e.g. 'monetary incentive delay'), allow examination of reward prediction, anticipation, and consumption (for review see (2; 3)). However, it has become apparent that reward processing is affected by whether an individual values being able to make their own choices: the inherent value of exercising personal control (4; 5). Being able to exert control over one's own environment is beneficial to psychological well-being (6), making investigations of such concepts relevant for patient groups and the wider population. Indeed, Self-Determination Theory (SDT) (7) argues that our core needs are for Autonomy (experience of enacting personal volition), competence (sense of mastery over one's environment) and relatedness (social belonging). These determine inclination to pursue behaviour for its own intrinsic enjoyment (8), which is at the heart of motivational anhedonia.

Derived from SDT is the concept of the 'locus of causality', describing the source from which a person perceives their behaviours to be motivated (9): (1) Autonomy-oriented individuals are intrinsically self-motivated, seeking out opportunities for information gathering, personal challenge and self-determination; (2) Control-oriented individuals take cues from environmental factors, e.g reward, deadlines and public opinion; (3) Impersonal-oriented individuals feel they have little intentional control over their behaviour, deferring to concepts such as luck or fate. Notably, the Impersonal orientation has previously been associated with depressive symptoms within healthy individuals (9).

Factor analysis suggests the causality orientations have partial overlap with personality concepts described by the NEO Five Factor Inventory: 'Control' shares variance with agreeableness, and 'Impersonal' with neuroticism; whereas 'Autonomy' stands as a separate entity (10). Moreover, whereas traits such as neuroticism are relatively stable over the life-course, and have a significant genetic underpinning (11), one's locus of causality is considered more dynamic (12), and is likely more environmentally-adaptive.

Neurobiologically, the feeling of personal control (13), even when illusory (14), is associated with striatal activation, which suggests it may itself incur an additional value signal not typically captured by reward-learning paradigms. Leotti & Delgado attempted to isolate this within a reward learning context by testing whether the mere anticipation of control, elicited by a cue signalling an opportunity to make a choice versus a passive selection, would recruit neural systems of reward. They found that cues indicating personal control elicited greater reward system activation in both reward-obtaining (15) and loss-avoiding (16) contexts. However, this previous paradigm did not clearly dissociate between choice anticipation and receipt of the reward itself. Here, we have adapted this 'value of choice' task to clearly separate anticipation and outcome phases of choice, and applied reinforcement learning models to better characterize the relationship between the value of choice and neural activation in healthy individuals.

Specifically, our aims were to: (a) verify previous findings concerning choice-anticipatory activation; (b) determine whether responses to rewards differ according to whether or not they were personally won or passively received; (c) establish that, with appropriate modification of the original paradigm, computational models of reinforcement learning can explain observed brain activity; (d) determine whether elicited activation co-varies with subclinical depressive symptoms, and personality factors relevant to depression,

namely neuroticism and measures of causality orientation. We anticipated that high neuroticism and Impersonal scores would be associated with diminished activation to the inherent value of choice because depression has been linked to other types of blunted reward value (17). We were particular interested in the roles that the striatum and dopaminergic midbrain might play, given their key importance in reinforcement learning, incentive salience and hedonic signalling.

Methods

Participants

Individuals were selected from a wider ongoing study ('STRADL', STratifying Resilience and Depression Longitudinally, (18)), and underwent lifetime diagnostic screening using the Structured Clinical Interview for DSM disorders (19) and Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) criteria. Only those without a lifetime diagnosis of major mental illness were included in the current analyses, which were performed when data from the first 149 healthy control participants were available. Exclusions comprised: fifteen people due to non-performance of the task (no response or incorrect for > 33% of trials); six due to scan acquisition technical difficulties; and six for excessive motion (>3 events involving motion > [0.5 x largest voxel dimension = 2.5mm]), leaving n=122 participants. All participants provided written informed consent, and the study was approved by local and regional ethics committees.

Neuropsychology and behavioural analyses

Neuropsychological data collected included the General Causality Orientations Scale (GCOS) (20), which examines the sources from which a person is motivated to act (9), consisting of 3 dimensions: Autonomy, Control and Impersonal. Neuroticism scores, the severity of depressive symptoms, and handedness were also assessed (see Supplementary Materials).

Neuroimaging data acquisition and preprocessing

Data were acquired using a 3T MRI scanner, TR=1.56s (see Supplementary Materials).

The modified inherent value of choice imaging task

The task was adapted from (15) and implemented in NeuroBehavioural System's Presentation software. Each trial had three phases (Figure 1): (1) the 'cue' phase, where participants learned whether they would personally be making the reward decision (Choice value trial), or would be following the computer's direction (No-choice value trial); (2) the 'selection' phase, whereby a decision was made between a yellow or blue card; and (3) the 'outcome' phase, when participants received a probabilistic reward according to their decision. During the selection phase, participants were able to freely select their preferred card during Choice trials; on No-choice trials, a rectangle appeared around the card which the computer had selected for them, which they were obliged to confirm. Selections were made via a button press.

In the original task, the yellow and blue cards shared equal reward contingencies. In our adaptation, they had different contingencies in order to permit modelling of reinforcement learning: the yellow card was associated with an 80% chance of a 100-point reward, and the blue card with a 20% chance. The alternative outcome was 0 points. We also introduced 1500-4000ms of jitter between selection and outcome phases of each trial, allowing for disambiguation of all three phases.

Participants completed 66 trials, 33 Choice, and 33 No-choice. Trial order and the side of the screen on which the yellow and blue cards appeared were randomised, preventing final action planning. Decisions made by the participant during Choice trials were mirrored by the computer with a 3-trial lag during No-choice trials, in an effort to match the overall rewards received across conditions of interest. Total task length was 14 min 59 sec.

Participants were told their objective was to learn which colour card was more likely to give them points, by trial and error. They were informed that for some trials they would get to choose, but in others the computer would chose for them. During the latter they had to follow the computer's selection. They were also told that the reward contingencies remained consistent regardless of whether they or the computer were doing the choosing. A post-scan questionnaire asked participants to rate their desire to win points on a scale of 1 to 10, and their preference for Choice or No-choice trials.

FMRI data analysis

Two analytic approaches were adopted:

1. The *basic model* was used to (i) verify that appropriate reward responses were seen for the outcome phase contrast of reward 100 > 0, regardless of Choice/No-choice; (ii) verify the results previously reported by (15) regarding cue phase Choice > No-choice activation; (iii) assess Choice > No-choice activation during the reward phase, and the Choice x reward interaction; and (iv) examine the associations between contrasts from (ii) and (iii) with the three GCOS causality orientations (Autonomy, Control, Impersonal), neuroticism and depressive symptomatology.
2. The *Pavlovian reward learning model* attempted use a computational framework to estimate how much each participant "valued" being able to choose, by fitting a temporal difference learning model to the data. This considered the cue phase Choice and No-choice indicators as though they were stimuli to be "conditioned" on subsequently obtained rewards. We proposed that the degree to which the model's

value estimate accounted for Choice anticipatory activation would be dependent on causality orientation, neuroticism and depressive symptomatology.

Basic model

This was modelled at the first level as a series of delta functions convolved with a canonical haemodynamic response function, the onsets of which were denoted by experimental conditions of interest. These were the onsets of the Choice and No-choice cues; and the onsets of trial outcome, with Choice/No-choice and 0/100 points being modelled separately, giving six experimental vectors of interest. Nuisance regressors included the onsets of yellow/blue selection; trials where an incorrect response or no response was received; and motion parameters.

At the second level, cue phase contrasts of Choice > baseline and No-choice > baseline were entered into a random-effects flexible factorial analysis, modelling the factors of participant and Choice/No-choice. The outcome phase was considered in a separate 2 x 2 flexible factorial analysis incorporating the contrasts of Choice 100 > baseline, Choice 0 > baseline, No-choice 100 > baseline and No-choice 0 > baseline, modelling the factors of participant, Choice/No-choice and reward amount (see Supplemental Methods). For both models, each participant's desire to win points, and any difference in points received for Choice versus No-choice trials, were included as nuisance covariates. Regions identified as showing significant activation for the contrasts of interest were subject to extraction of the first eigenvariate for the suprathreshold cluster, and their relationships with our covariates of interest explored (Autonomy, Control, Impersonal, QIDS depressive symptoms and EPQ-R neuroticism scores). This was done using backwards regression within SPSS (version 23, https://www.ibm.com/analytics/us/en/technology/spss/): for each extracted region, the

model that best accounted for the data was identified by ANOVA; within this, significant coefficients of explanatory covariates being reported. These were subjected to FDR correction with q=0.05 across all comparisons, and standardised β values reported.

Pavlovian reward learning model

The task was also modelled as an instance of classical conditioning, using a temporal difference learning model (21). We wished to identify whether learning rate varied according to whether or not participants were actively choosing. The model implemented four different learning rates: 0.2, 0.4, 0.6 and 0.8, used to generate cue value and prediction error (PE) estimates across the task for each participant, based on their cue-outcome experiences during the scan. The unconditioned stimulus (US) was the outcome phase of each trial (the receipt of 100 or 0 points). The conditioned stimuli (CS) were the Choice and No-choice indicators during the cue phase (see Supplementary Materials). Cue value was used to modulate trial-by-trial regressors representing the cue phase of each trial, and PE was modulated the outcome phase. Choice and No-choice conditions were modelled separately. These were entered into first-level SPM analyses, with a different SPM for each learning rate. Contrast estimates for each regressor were taken into second-level 2 x 4 flexible factorial analyses, which modelled the main effects of participant, Choice/No-choice and learning rate. As we expected choice value estimates to strongly covary with measures of Autonomy, Control, Impersonal, neuroticism and depression scores, these were included in the second-level analyses, modelling interactions with both Choice/No-choice and learning rate.

For both the Basic and Pavlovian models, second-level contrasts were evaluated at a whole-brain voxel height threshold of $p<0.05$ FWE-corrected. Given *a priori* interest in the striatum and dopaminergic midbrain, we also conducted region-of-interest analyses within a structurally-defined mask comprising bilateral caudate, putamen, and dopaminergic midbrain (see Supplementary Materials). Masked voxels were reported as significantly activated if they exceed a FWE-corrected height threshold of $p<0.05$.

Results

Demographics, neuropsychology and symptoms

Median age was 62 years; and 46% were male (Table 1). There was no correlation between age and task performance ($p > 0.823$). 93% of participants preferred making their own choices. Learning continued throughout the task, with the most rewarding card being chosen 79% of the time during the final quarter of the session (Supplemental Figure 3). Both QIDS depression ($\tau=0.213$, $p=0.003$) and Impersonal scores ($\tau=0.197$, $p=0.003$) were positively correlated with neuroticism.

Basic model: (i) Reward verification

See supplementary materials.

Basic model: (ii) Verifying anticipation of choice: cue phase

Cue phase Choice > No-choice revealed strong activation in the cerebellum, left insula, left cingulate/SMA, and right IFG, corrected for the whole brain volume (

Figure 2, Table 2). Bilateral putamen was activated within the striatum/midbrain *a priori* mask (Figure 3a). No-choice > Choice showed activation in occipital cortex only (Table 2).

Basic model: (iii) Reward and choice: outcome phase

Next we examined whether responses to personally-earned outcomes differed from those passively received. The outcome phase Choice > No-choice contrast showed significant activation in the bilateral insula, anterior cingulate, right IFG, left hippocampus and left thalamus (

Figure 2, Table 3). Within the striatum/midbrain ROI, there was significant Choice > No-choice activation within the left substantia nigra and right caudate nucleus. No-choice > Choice activated left middle frontal cortex, precuneus and angular gyrus.

Reward x choice interaction activations

The contrast of Choice (0 > 100) > No-choice (0 > 100) showed activation in right IFG pars opercularis (Table 3). Conjunction analysis confirmed that this lay within the Choice > No-choice cluster (P=0.038 FWE-corrected), but not that of 0 > 100 (**Error! Reference source not found.**a). Contrast estimates suggested enhanced activation when one personally failed to win (**Error! Reference source not found.**b).

Relations to traits of interest

QIDS depressive symptoms were negatively related to left putamen anticipation: Choice > No-choice (β=-0.365, p<0.001, Figure 3b). During the outcome phase, Autonomy had a positive association with right IFG/insula Choice > No-choice activation (β=0.280, p=0.045), whereas Impersonal demonstrated the inverse relationship (β=-0.255, p=0.025). Within precuneus, Control had a positive relationship during outcome: No-choice > Choice activation (β=0.396, p<0.001), but conversely Impersonal showed a negative association in the same region (β=-0.288, p=0.012, Supplemental Figure 4). Here QIDS depression demonstrated a similar pattern to Control (β=0.226, p=0.039). Table 4 details these relationships.

<u>Pavlovian reward learning: The value of personal choice</u>

The final analytical thread considered whether the ability to choose was intrinsically rewarding in itself, within a reinforcement learning context. During the cue phase of each trial, there were no main effects of Choice/No-choice or learning rate. However as anticipated there was significant covariation with several metrics of interest (Table 5). Increasing Autonomy was associated with greater No-choice > Choice value estimates in right amygdala ($p=0.008$), and greater Choice > No-choice value estimates in anterior caudate ($p=0.019$). Again during the cue phase, Control orientation demonstrated a positive relationship with learning rate in the right superior temporal sulcus ($p=0.017$).

During the outcome phase, there was a significant main effect of learning rate (α) in ventral striatum, with a lower α being associated with greater PE representation ($p<0.001$). Conversely, there was an effect of increasing α in the right anterior insula and SMA ($p<0.006$). Learning in ventral striatum therefore appears to operate over a longer timescale than in insula/SMA. Finally, Impersonal showed a stronger PE representation for No-choice > Choice in bilateral nucleus accumbens ($p<0.004$, Figure 3c and d). For interest, results significant at $p < 0.001$ uncorrected can be found in the supplemental materials.

Discussion

In this study, we modified Leotti and Delgado's 2011 inherent reward of choice task, to (a) verify their previous findings; (b) disambiguate the cue and outcome phases; (c) demonstrate the utility of computational models in this context; and (d) see whether task-elicited activation covaried with personality factors of relevance to depression. Their findings concerning choice anticipation were replicated within our larger independent sample of healthy controls. The task was amenable to Pavlovian reward learning analysis. We then demonstrated a series of novel findings within regions key to reward and depression, their relation to depressive symptoms, and measures that attempt to "personalise" notions of reward and value. This aligns them with the depressive phenomena of motivational anhedonia, and devaluation of the self.

Anticipating choice

We verified the striatal anticipatory response to choice as seen in Leotti and Delgado, 2011. Critically we observed that this effect was diminished in those with more depressive symptoms, suggesting an impairment in the hedonic value or salience attributed to personal choice. Reduced ventral striatal reward-linked responses are a well replicated finding in those with MDD, be it when viewing positive images (22) - which correlates with anhedonia (23) - or anticipating and receiving rewarding outcomes (24; 25). In healthy controls, depressive symptoms correlate with a reduction in the usual performance-enhancing effects of positive feedback, implying striatal dysfunction (26). Striatal activation correlates with enhanced recall of personally-chosen items, and exerts a modulatory effect over hippocampus (27): this mechanism may underpin the cognitive biases observed in MDD. It is notable that we too report striatal dysfunction in a group of healthy controls, who have

not been subject to the effects of medication or an episodic illness, while having a narrower distribution of depressive symptoms. We also find enhanced insula and cingulate activation during Choice > No-choice anticipation: these regions have been shown to correlate with momentary subjective well-being in rewarding contexts (28), supporting the view that personal choice is intrinsically appetitive. Both are key components of the salience network, and play a role in cognitive control (29).

Personally-earned versus passively-received rewards

Responses to personally-chosen outcomes were enhanced compared to those that were passively received: insula/IFG and cingulate cortex were apparent, as were hippocampus, thalamus and substantia nigra. Right IFG pars opercularis (IFGpo) demonstrated a choice x reward interaction, whereby there was an enhanced response when participants personally failed to win. Right IFGpo plays a specific role (30) in the inhibition of motor and affective responses (31). It is also activated by personal "regret" versus simple disappointment (32). It could be argued that personally failing to win induces a self-blame response (33) that requires inhibition or emotional regulation. Such a response would be relevant to depression and particularly to resilience in the face of adversity (34).

Inferior frontal gyrus and goal-sensitive self-regulation

Right IFG/insula showed a Choice > No-choice response across the sample during the outcome phase, which was enhanced by high Autonomy, but diminished by high Impersonal scores. The concept of locus of causality is not far removed from that of learned helplessness, which inspires animal model of MDD, and gives ventral prefrontal cortex (IFG) particular prominence in a recent update by its architects Maier and Seligman (35).

Prolonged aversive events are proposed to stimulate the raphe nuclei, releasing serotonin within the striatum (inhibiting behaviour) and amygdala (inducing fear and anxiety), irrespective of detected contingencies. This response is inhibited if the agent has previous experience of acting to escape aversive events, mediated by ventral prefrontal cortex's regulatory influence over the raphe nuclei and striatum. They suggest this process equates to the agent being able to imagine having control over future aversive situations. Right IFG/insula are crucial contributors to cognitive control, governing the ability to select and maintain goal-directed action at the expense of other alternatives (36). Strong meta-analytic evidence support their role in the cognitive reappraisal of emotional stimuli (37). Reduced responses to negative affective stimuli have been reliably demonstrated in those with MDD (38). Here we show the IFG's response to personal choice is greater in those having high Autonomy, and reduced in those having an Impersonal, passive style. The latter may therefore have a reduced ability to act to escape aversive situations, and regulate subcortical limbic responses to aversive events, whereas the former would be more adaptive and resilient. Bhanji and colleagues linked resilience to believing one has personal control (39): they found that one's ability to overcome setbacks was reduced following exposure to an acute stressor; however this was diminished in those who believed that they had some control over the setbacks.

The precuneus and agency perception

The precuneus showed a No-choice > Choice response during the outcome phase, especially so in those with high Control, with the opposite being seen with high Impersonal scores. Precuneus is part of the default mode network, and generally deactivates during goal-directed tasks (40). This happens to a lesser degree during tasks having a self-referential

component, taking a first person perspective, or inducing the experience of agency (41). It also activates when mentally simulating the actions of another versus oneself (42), taking perspectives alternative to one's own (43), and considering the emotional states of both yourself and others versus neutral judgements (44). More abstractly, it is activated during judgements of intentional versus simple physical causality (45). In summary, it is arguable that any process that involves consideration of an intentional agent engages precuneus, regardless of whether this is one's own self, although the self is likely to prevail during default mode operations. The Control orientation may increase the propensity to seek cues in the minds of others, and consider the computer's "intentions". Conversely, the Impersonal orientation show an apparent abolition of the effect seen in the general sample, suggesting a reduced inclination to consider intentionality at all.

Reinforcement learning

The final analysis phase attempted to capture the learning process underlying how the Choice/No-choice cues developed their inherently rewarding character, and how this related to participants' characteristics. The use of a computational model potentially allows for a more mechanistic understanding of the activation observed, and highlighted relationships with personality metrics that weren't detected during the basic analysis. Highly Autonomous people encoded value during presentation of the No-choice cue within right amygdala, suggesting that either No-Choice cues (46), or the uncertainty associated with what the computer might select (47), were regarded as aversive. They also showed greater Choice > No-choice cue valuations in dorsal anterior caudate, which through its interactions with prefrontal cortex plays a crucial role in goal-directed action (48). High Control participants showed enhanced learning in the right superior temporal sulcus, which is

especially involved in considering the intentions of external others (49). Finally, high Impersonal participants had stronger nucleus accumbens PE signals for passively-received rewards, suggesting that a reduced belief in the ability to control one's behaviour related to more reward system reactivity to "gifted" versus "earned" rewards.

Limitations

A number of study participants were unable to perform the task correctly, suggesting that it was subjectively hard to understand, or that a potentially important section of the population have been excluded. We have not examined trial-by-trial assessments of choice preference, or changes in stay/switch behaviour, which are also believed to covary with depressive symptoms (26). The 80:20 yellow:blue reward contingency was used to permit reliable learning across a range of participants, and may have induced ceiling effects in some participants, as we did not find a simple interaction between Choice/No-choice and learning rate. However it allowed us to focus on whether or not the participant did the choosing, without that choice in itself being particularly onerous. Indeed, if a choice versus no-choice decision involved a difference in deliberation that could have introduced additional confounds. Alternatively, our temporal difference learning model may not have adequately captured the variance introduced by personal choice.

Clinical relevance

Our findings suggest that the modified inherent value of choice task could be able to provide useful insights into the neurobiology of MDD. Within this large sample of healthy controls, we have shown how personal choice modulates activation within areas known to be disrupted in MDD. This covaries with how inclined a participant is to see themselves as

the driver of their actions; to look to the outside world for their cues; or even to feel at a loss as to why they act at all. Being able to tease apart how particular manifestations of personality impact on one's vulnerability to MDD is likely to be important to stratification. Characteristics such as causality orientation arguably build on more stable and heritable measures such as neuroticism, as they are more responsive to environmental events, and so may provide more timely information regarding the risk of transition to illness, as well as offering targets for psychotherapeutic interventions. The hope is that by examining the reward system in a manner that ties self-perception to behaviour, more clinically-applicable insights can be drawn. For example, a particularly effective therapeutic strategy for those having a high Impersonal/low Autonomy style might be to both enhance dopaminergic transmission, and challenges self-orientation beliefs during CBT.

Tables

				Kendall's τ Correlation (p)
Measure (possible range)	**Median (IQR)**	**Skewness**	**Kurtosis**	**Neuroticism**
Age	62.0 (3.00)	-0.574	0.630	
Sex (F:M)	56:66			
Handedness (R:L:A)	111:5:6			
GCOS: Autonomy (12-84)	68 (10)	-0.881	1.071	
GCOS: Control (12-84)	48 (10)	-0.007	0.637	
GCOS: Impersonal (12-84)	37 (16)	-0.176	-0.714	0.197 (0.003)
Neuroticism (0-12)	2 (3)	1.217	1.843	
QIDS (0-27)	3 (2)	1.262	2.382	0.213 (0.003)
Desire to win (1-10)	7.5 (4.0)	-0.742	-0.047	
Trial preference (Choice:No-Choice)	114:9			
Points won (0-6600)	3500 (1200)	-0.384	0.017	
No-Choice / Choice points	0.623 (0.39)	-0.044	-1.167	
No-choice trials missed (0-33)	1.00 (3.00)	1.840	2.675	

Table 1: Demographic, personality, symptom and behaviour measures. Only significant correlations between personality and symptom measures are shown. No-choice trials where the participant chose the card not pre-selected by the computer were defined as "missed". IQR is interquartile range. Standard error of skewness was 0.220, and of kurtosis was 0.437.

Contrast	Region	MNI coords	Voxels	T	Z	P (FWE-corrected)
Choice > No-choice	L cerebellum	-38 -56 -52	55	5.57	5.24	0.001
	L insula	-40 16 2	63	5.29	5.01	0.004
	R IFG	50 12 6	24	5.06	4.81	0.010
	L cingulate/ SMA	-10 28 32	20	5.04	4.80	0.010
	R insula	40 18 6	5	4.68	4.48	0.038
	L putamen	-20 10 -2	84	4.33	4.17	0.008*
	R putamen	22 12 -4	55	4.19	4.04	0.012*
No-choice > Choice	R occipital cortex	12 -88 -2	118	6.56	6.05	<0.001
	L occipital cortex	-18 -82 -14	148	6.11	5.69	<0.001

Table 2: Choice/No-choice anticipatory activation during the cue phase. FWE-corrected p values are for the whole brain volume, except where "*" denotes FWE-corrected significance within the striatum/midbrain mask. IFG: inferior frontal gyrus. SMA: supplementary motor area.

Contrast	Region	MNI coords	Voxels	T	Z	P (FWE-corrected)
Choice > No-choice	R insula/IFG	36 20 -8	1789	8.93	Inf	<0.001
	R cingulate/medial superior frontal	0 38 28	3125	7.78	7.47	<0.001
	L insula	-36 20 -10	303	6.05	5.90	<0.001
	L ventral anterior thalamus	-12 -2 -6	34	4.69	4.62	0.024
	L hippocampus	-26 -6 -16	4	4.60	4.53	0.034
	L substantia nigra	-8 -14 -14	122	4.45	4.38	0.003*
	R caudate	12 4 8	4	3.85	3.81	0.030*
No-choice > Choice	L MFG	-30 30 52	75	5.40	5.29	0.001
	L precuneus	-2 -64 32	36	4.81	4.73	0.015
	L angular gyrus	-48 -62 24	27	4.80	4.72	0.015
Choice (0 > 100) > No-choice (0 > 100)	R IFG	34 20 10	5	4.56	4.50	0.038

Table 3: Outcome phase activation: Choice versus No-choice. FWE-corrected p values are for the whole brain volume, except where "*"denotes FWE-corrected significance within the striatum/midbrain mask. IFG: inferior frontal gyrus. MFG: middle frontal gyrus. MTG: middle temporal gyrus.

Phase	Region	Contrast	Metric	Standardised β	T (DoF)	P (corrected)
Cue	L putamen	Choice > No-choice	QIDS	-0.365	3.734 (113)	<0.001
Outcome	R IFG/insula	Choice > No-choice	Autonomy	0.280	2.253 (113)	0.045
			Impersonal	-0.255	2.758 (113)	0.025
	L precuneus	No-choice > Choice	Control	0.396	4.888 (114)	<0.001
			Impersonal	-0.288	2.986 (115)	0.012
			QIDS	0.226	2.467 (113)	0.039

Table 4: Relationships between significant activation clusters and metrics of interest (GCOS causality orientation, neuroticism and QIDS depressive symptoms). DoF: degrees of freedom.

Phase	Contrast	Region	MNI	Voxels	T	Z	P (FWE-corrected)
Cue (CS) x model value	Autonomy x (No-choice > Choice)	R basolateral amygdala	26 0 -24	14	4.72	4.69	0.008
	Autonomy x (Choice > No-choice)	L dorsal anterior caudate	-18 18 8	20	4.49	4.46	0.019
	Autonomy x Decreasing α	L anterior caudate	-18 26 4	32	4.72	4.70	0.003
	Control x Increasing α	R superior temporal sulcus	48 -24 -6	31	4.52	4.49	0.017
Outcome (US) x model prediction error	Main effect: Decreasing α	B ventral striatum	-12 10 -12	51	5.81	5.76	<0.001
	Main effect: Increasing α	R insula	-34 20 8	37	5.09	5.05	0.005
	Main effect: Increasing α	L supplementary motor area	-4 22 44	132	5.29	5.24	0.002
	Impersonal x (No-Choice > Choice)	B nucleus accumbens	-12 16 -10	23	4.44	4.41	0.004*

Table 5: Pavlovian conditioning of Choice versus No-choice. CS: conditioned stimulus. US: Unconditioned stimulus. X denotes an interaction between a GCOS subscale, and the contrast described. FWE-corrected p values are for the whole brain volume, except where "*"denotes FWE-corrected significance within the striatum/midbrain mask.

Figure captions

Figure 1: The modified inherent value of choice task

Figure 2: Choice > No-choice activation during the cue (red) and outcome (green) phases. (a) L cingulate cortex, (b) L hippocampus and thalamus, (c) Anterior insula and inferior frontal cortex, and (d) L subtantia nigra. Images shown achieve a whole-brain voxel height significance threshold of $p < 0.05$ FWE-corrected.

Figure 3: (a) Choice > No-choice anticipation (Cue phase) contrast demonstrating activation in bilateral putamen. (b) The relationship between QIDS depression score and L putamen activation (β=-0.365, $p<0.001$). (c) Left nucleus accumbens shows a correlation between Impersonal orientation and No-choice > Choice effect for Outcome phase prediction error encoding. (d) The relationship between Impersonal orientation and L nucleus accumbens Outcome phase prediction error activation. For (a) and (c), results achieve a voxel-height significance of $p < 0.05$ FWE-corrected within the striatum/midbrain mask.

Figure 4: The choice x reward interaction within R IFG during the outcome phase. (a) Activation maps for Choice > No-choice (red), reward 0 > 100 (green) and the choice x reward interaction (blue), displayed at a whole brain voxelwise FWE-corrected threshold of $p < 0.05$. (b) Contrast estimates extracted from the interaction cluster.

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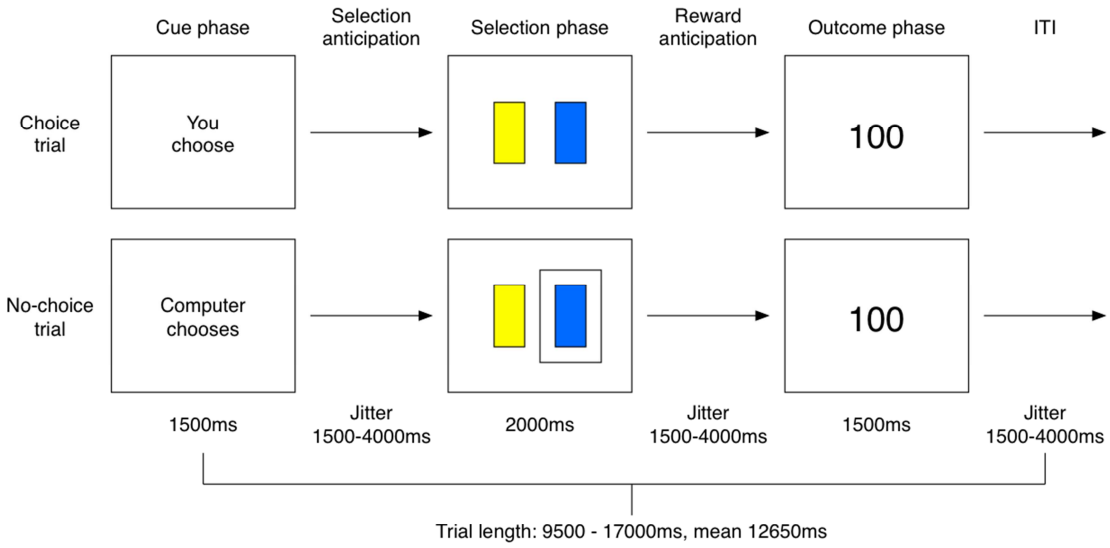

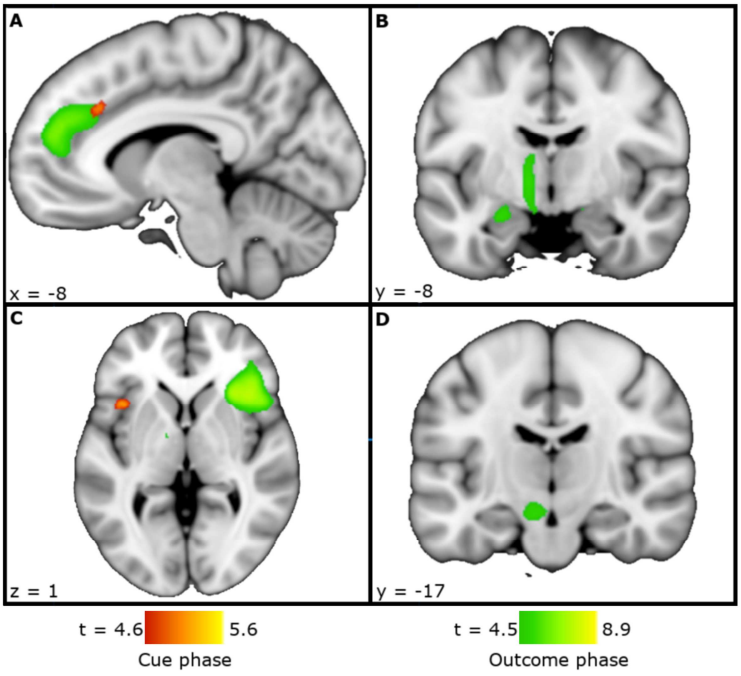

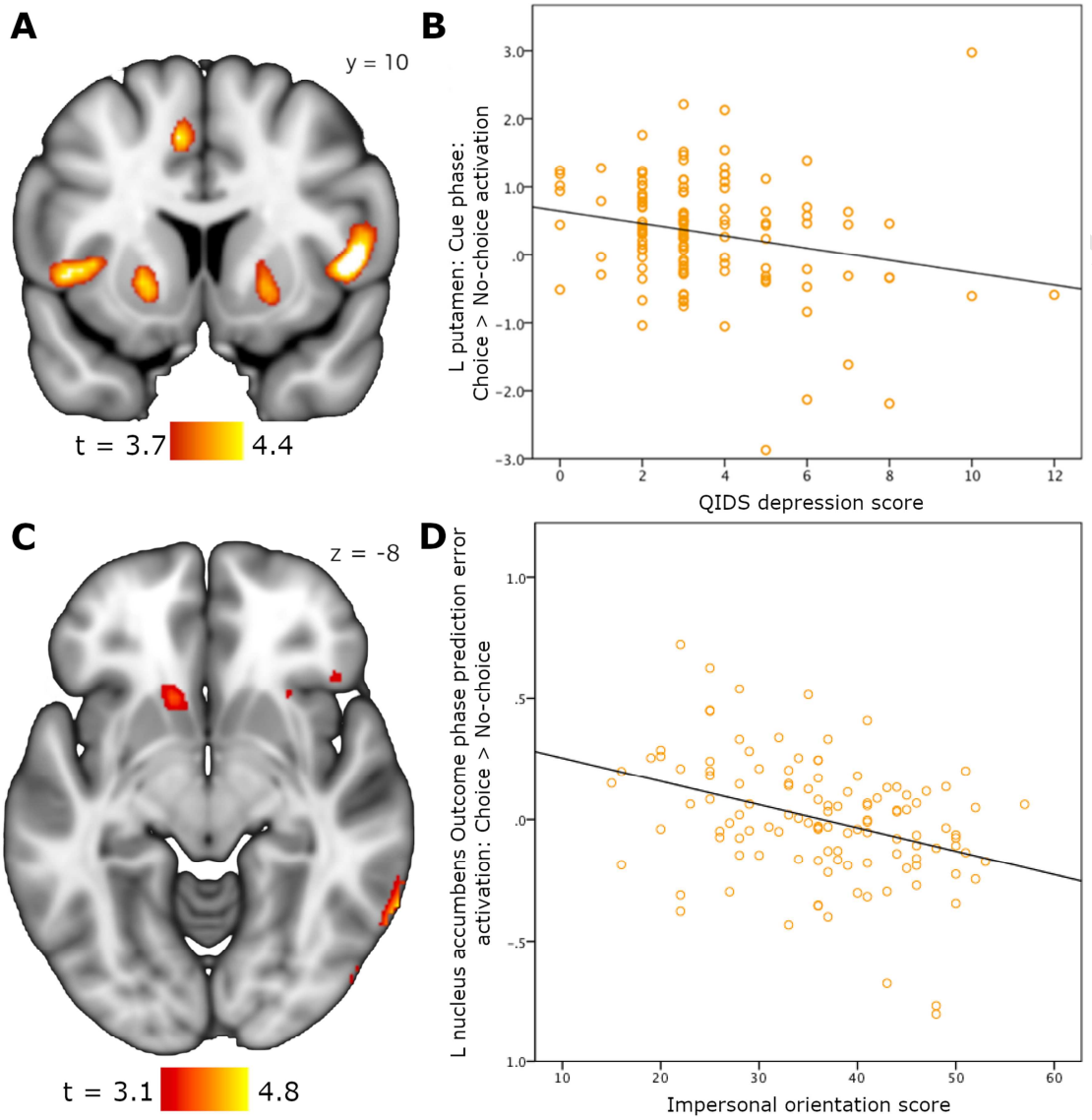
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t = 3.7
4.4
B
L putamen: Cue phase:
Choice > No-choice activation
3.0
2.0
1.0
.0
-1.0
-2.0
-3.0
0
2
4
6
8
10
12
QIDS depression score
C
z = -8
t = 3.1
4.8
D
L nucleus accumbens Outcome phase prediction error
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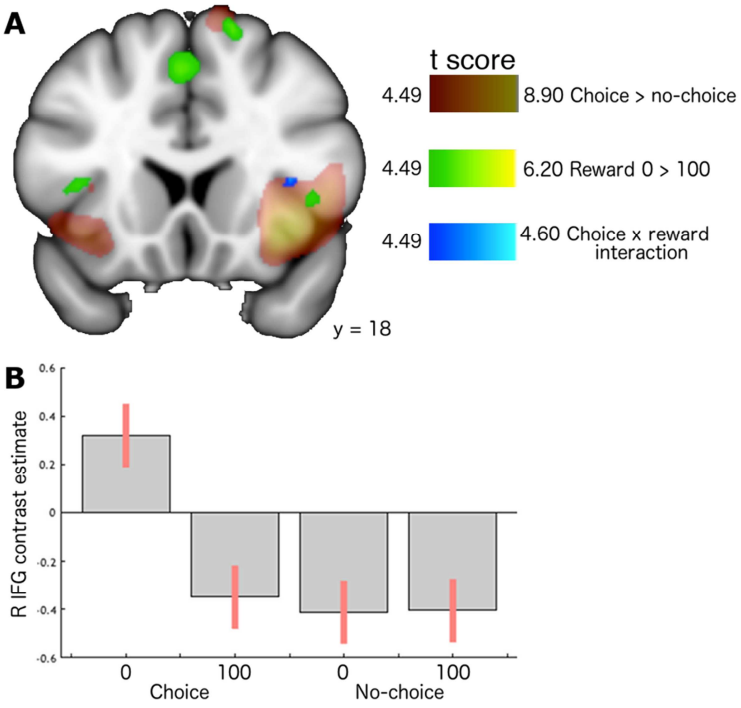
Impersonal orientation score



The Neurobiology of Personal Control During Reward Learning and Its Relation to Mood

Supplemental Information

Neuropsychology and behavioural analyses

The General Causality Orientations Scale (GCOS), is derived from Self-Determination Theory (1), examines the sources from which a person is motivated to act (2), consisting of 3 dimensions: Autonomy, Control and Impersonal. Participants rate 12 vignettes of situations probing these dimensions, with total subscale scores ranging between 12 and 84. Neuroticism scores were derived from the Eysenck's Personality Questionnaire – Revised (EPQ-R; short form (3)). The severity of depressive symptoms were derived by self-report using the Quick Inventory of Depressive Symptomology (QIDS; (4)). Handedness was determined using the Edinburgh Handedness Inventory (5).

Neuroimaging data acquisition and preprocessing

Data was acquired using a Philips Achieva 3T TX-series scanner (Philips Healthcare, Best, Netherlands) at the University of Aberdeen, with a 32-channel phased-array head coil with a back-facing mirror (software version 5.1.7; gradients with maximum amplitude 80 mT/m and maximum slew rate 100 T/m/s). A projector and "Presentation" (Neurobehavioural Systems) version 18.1 were used for the presentation of task based fMRI.

fMRI data were acquired with a TR = 1.56s and TE = 26ms. FA = 70', FOV = 217mm, matrix size = 64 x 64. In-plane resolution was 3.4 x 3.4mm, with 32 5mm axial slices being acquired continuously with no gap. 573 volumes were collected, with the first six being discarded to

accommodate T1 saturation effects. A T1-weighted structural image was acquired as 160 sagittal slices, with TR = 8.3ms, TE = 3.8ms, TI = 1031ms, FA = 8', FOV = 240mm, matrix size = 240 x 240, giving a resolution of 0.9 x 0.9 x 1.0mm. Data were preprocessed and analysed using SPM12 (Wellcome Department of Imaging Neuroscience, London, England; http://www.fil.ion.ucl.ac.uk/spm/software/spm12/), within MathWorks MATLAB R2016a (http://www.mathworks.com). fMRI volumes were reconstructed into NIfTI format, and realigned to the mean volume. The structural image was segmented and warped to MNI space. The mean fMRI and structural volumes were coregistered, and the normalisation parameters applied to the whole fMRI dataset which was then smoothed using an 8mm FWHM gaussian kernel, and resampled at 2mm isotropic resolution. The data was high pass filtered with 128s cutoff, and serial correlations modelled using a first-order autoregressive model.

Basic model flexible factorial analysis

For both the cue phase and outcome phase second level analyses, the factor of participant was modelled as having independent and equal variance, as it was not anticipated that the healthy control population from which the sample was drawn would demonstrate wide variance between individuals. The factor of Choice/No-choice was also modelled for both these analyses, this time having dependent and equal variance, as this was a within-subjects comparison. The outcome phase analysis included the additional factor of reward amount (100 or 0 point): this was again modelled as dependent and equal variance, for the same reasons as Choice/No-choice.

Pavlovian reward learning methods

Each trial was modeled over six time points, with the CS occurring at t = 1, and US at t = 3. The predicted value for each CS was calculated at each time point:

$$\hat{\boldsymbol{V}}(t)=\boldsymbol{w}\boldsymbol{x}(t)$$

Where V, w and x are vectors with separate entries for the Choice and No-choice indicators. X(t) is a binary vector denoting the presence of each Choice/No-choice indicator and w is the learned weight accorded to each CS. The predicted value V is updated at each time step according the PE, that is, to the difference between its estimation at the current time step, and the next:

$$\delta(t) = r(t) + \lambda \hat{\boldsymbol{V}}(t+1) - \hat{\boldsymbol{V}}(t)$$

Where r(t) is the outcome at time t. This is 1 when 100 points are received, and 0 otherwise. λ is a temporal discounting factor, which was set to 1. Weights were updated for each trial according to:

$$\Delta w = \alpha \sum_{t} x(t)\delta(t)$$

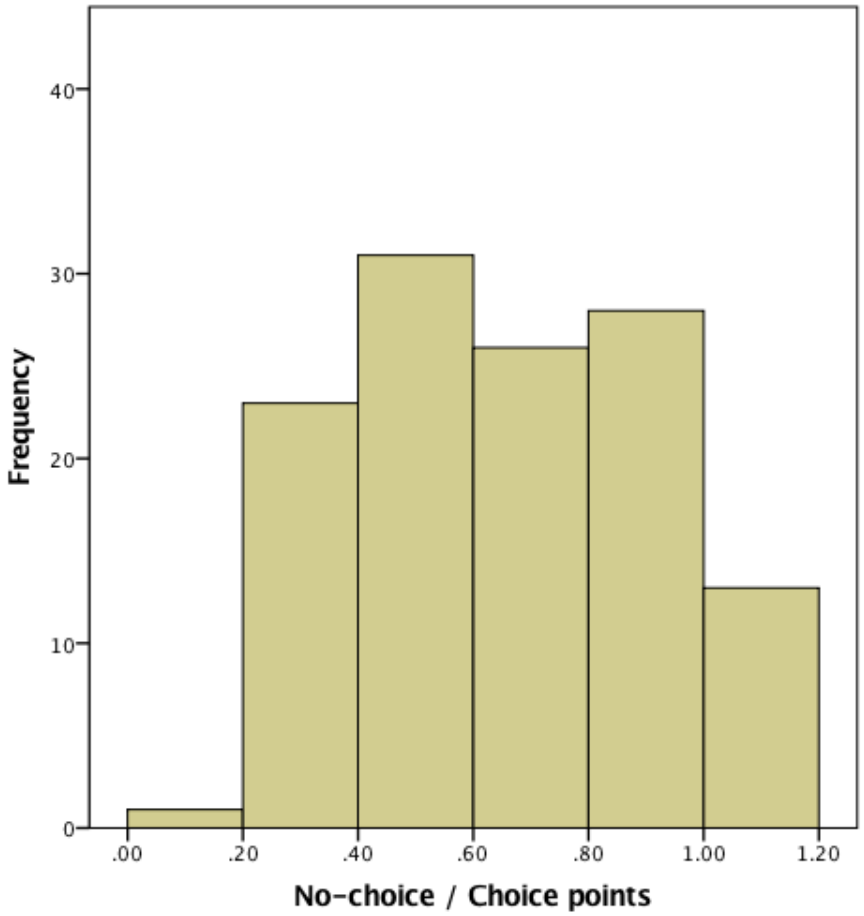
Where α is the learning rate.

Functional imaging mask

The striatal/dopaminergic midbrain mask was created from a union of Automated Anatomical Labeling-defined caudate and putamen; Brodmann-defined substantia nigra; and a 10mm sphere centred on the ventral tegmental area at MNI coordinates 0 -20 -10 (6), using the WFU Pickatlas (7; 8).

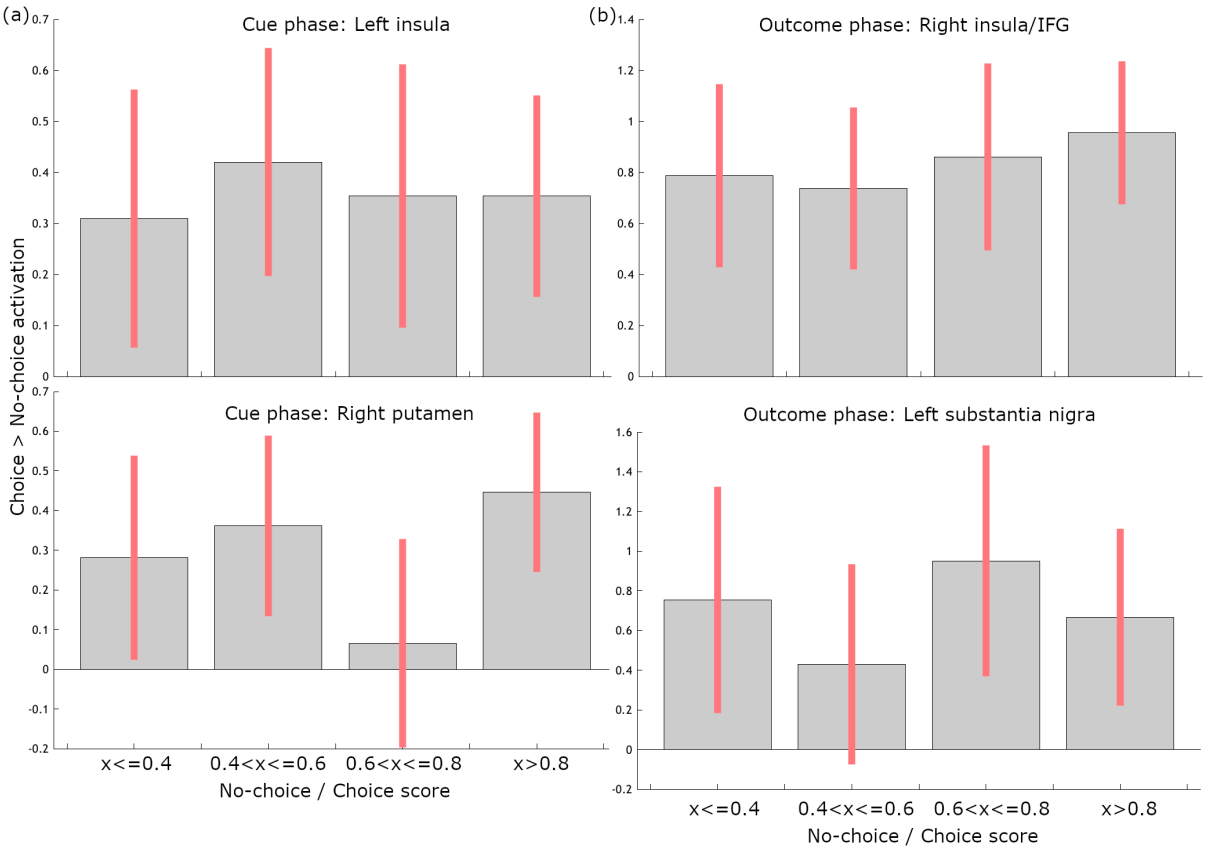
Behavioural results

For a number of our participants, it was found that a random run of No-choice trials would lead to there being a relatively insufficient number of Choice trials for the computer to copy the behaviour of. In these circumstances, the system would default to forcing the participant to select the losing card during No-Choice trials. This led to some of our participants receiving a lower number of points for No-choice relative to Choice trials (Supplemental Figure S1). This issue had no correlation with age, QIDS depression, neuroticism or causality orientation metrics ($p > 0.208$). Point difference was included as a nuisance regressor in all the imaging analyses described here. We also performed an additional series of analyses where an additional factor of Group was modelled whereby participants were divided into independent groups with No-Choice/Choice scores <= 0.4, 0.4 < scores <= 0.6, 0.6 < scores <= 0.8 and scores > 0.8. The interaction between score group and Choice/No-choice was examined. Contrast estimates were also extracted to demonstrate how activation varied according to Choice and score group.



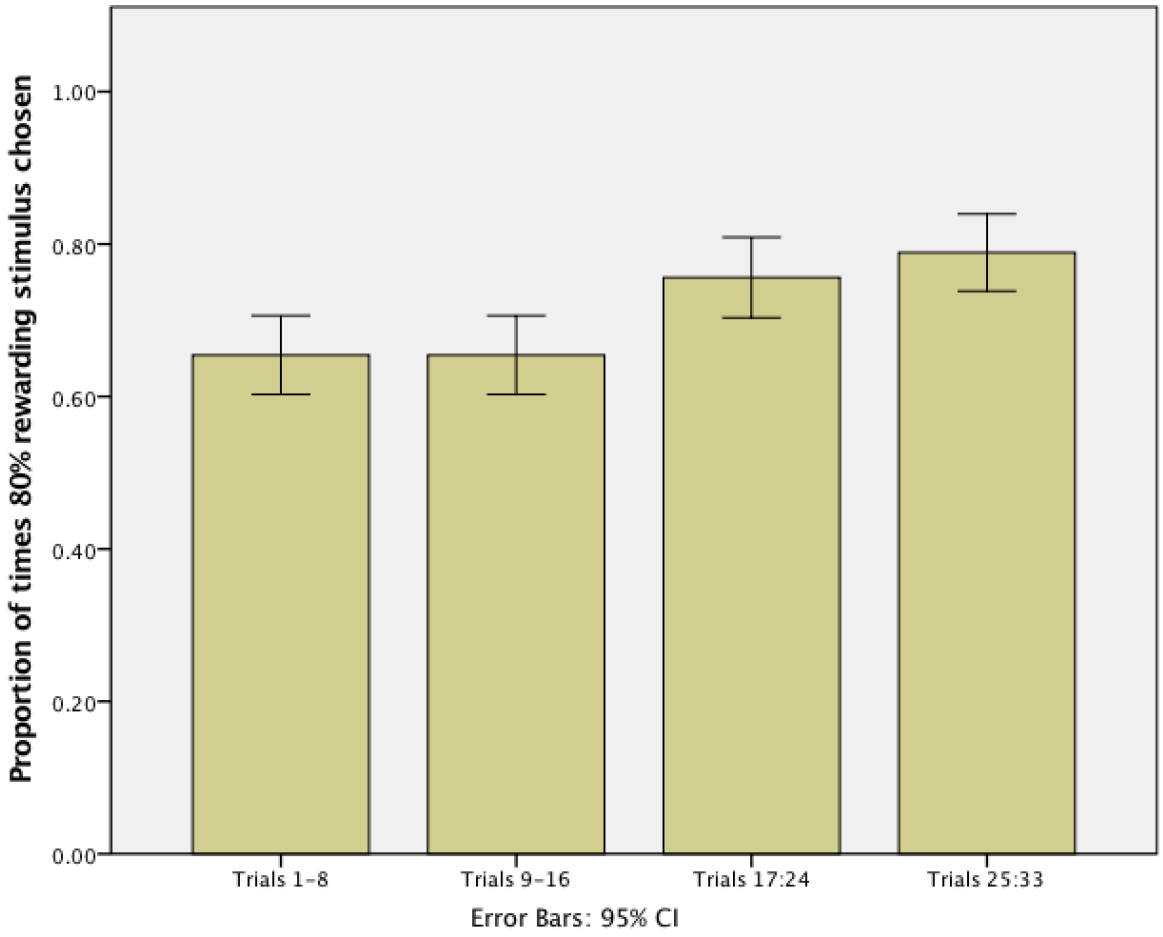
Supplemental Figure S1: The distribution of the difference in points for Choice and No-choice trials.

During the Cue phase for the basic analysis (analogous to Table 1 in the main paper), there were no significant interactions between Choice/No-choice and score group ($P > 0.246$). Representative extracted contrast estimates for left insula and right putamen are shown in Supplemental Figure S2(a), which suggest that the Choice > No-choice effects reported are not attributable solely to point differences.



Supplemental Figure S2: (a) Cue phase contrast estimates demonstrating the apparent lack of impact of point differences on Choice > No-choice activation in two key regions reported in Table 2. (b) Outcome phase contrast estimates for two regions reported in Table 3.

Likewise, for the Outcome phase, there were no score group x Choice/No-choice interactions ($p > 0.432$). Supplemental Figure S2(b) provides representative contrast estimates.



Supplemental Figure S3: Proportion of times the yellow (most rewarding) card was chosen during Choice trials.

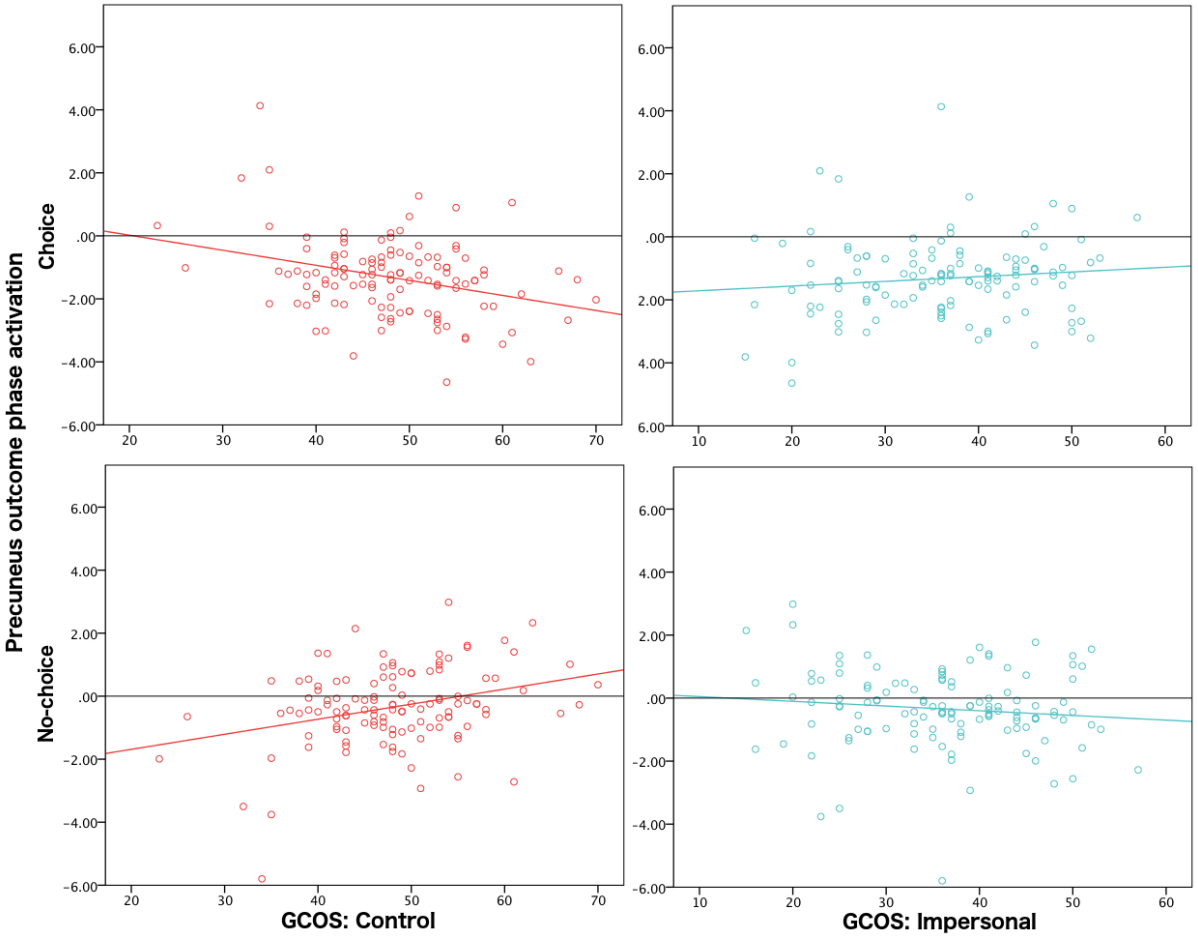
Basic model results: Outcome phase, reward 100 <> 0

The main effect of reward 100 > 0 across both the Choice and No-choice conditions revealed robust activation within regions previously associated with typical reward responses, such as the nucleus accumbens and orbitofrontal cortex. The inverse contrast of 0 > 100 showed right angular gyrus, left supplementary motor area (SMA) and bilateral insula/inferior frontal gyrus (IFG) activation (Supplementary Table S1).

Supplemental Table S1: Outcome phase activation: reward 100 versus reward 0.

Contrast	Region	MNI coords	Voxels	T	Z	P (FWE-corrected)
Reward 100 > 0	L + R occipital cortex	16 -94 10 -14 -96 0	13832	29.17	Inf	<0.001
	L precentral gyrus	-52 0 50	223	7.75	7.45	<0.001
	L nucleus accumbens	-10 8 -12	102	6.42	6.24	<0.001
	L STG	-56 -6 -12	200	6.42	6.24	<0.001
	R medial SFG	6 54 -8	504	5.56	5.44	0.001
	R ventral putamen	14 8 -8	4	5.13	5.04	0.004
	L orbitofrontal cortex	-24 32 -16	16	4.72	4.65	0.021
Reward 0 > 100	R angular gyrus	50 -56 56	998	6.15	5.99	<0.001
	L SMA	-4 22 46	252	5.30	5.20	0.002
	R SFG	16 18 62	53	5.04	4.95	0.006
	L insula/IFG	-38 20 -8	25	4.90	4.82	0.010
	R insula/IFG	44 18 4	12	4.76	4.69	0.018

FWE-corrected p values are for the whole brain volume. IFG: inferior frontal gyrus. SFG: superior frontal gyrus. SMA: supplementary motor area. STG: superior temporal gyrus.



Supplemental Figure S4: Precuneus outcome activation in relation to Control (β = 0.396, p < 0.001) and Impersonal scores (β = -0.288, p = 0.012).

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